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L1 374 S EF-2  
L2 16705 S NEURODEGENER?  
L3 0 S L1 AND L2  
L4 878553 S NEURO?  
L5 11 S L1 AND L4  
L6 4177 S ELONGATION FACTOR  
L7 4259 S L6 OR L1  
L8 43 S L7 AND AD  
L9 56 S DIPHTHAMIDE?  
L10 0 S L9 AND NEURO?  
L11 0 S L9 AND AD  
L12 7 S L2 AND L7  
L13 13283 S DIPHTHERIA?  
L14 2 S L13 AND L2  
L15 306 S L13 AND L4

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L16 1575 S L13 AND L2  
L17 144 S L16 AND L6  
L18 583 S L13 AND ELDERLY  
L19 434 S L18 AND NEURO?  
L20 153 S L19 AND DEMENTIA

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**Research report****Alzheimer's disease-associated reduction of polysomal mRNA translation**N. S. Langstrom<sup>b, a</sup>, J. P. Anderson<sup>a</sup>, H. G. Lindroos<sup>c</sup>, B. Winbland<sup>b</sup> and W. C. Wallace<sup>a</sup>, <sup>a</sup> The Mack Laboratory, Department of Psychiatry and Arthur M. Fishberg Center for Neurobiology, Mount Sinai School of Medicine, New York, NY, U.S.A.<sup>b</sup> Department of Geriatric Medicine, Karolinska Institute, Huddinge, Sweden<sup>c</sup> Department of Physiological Chemistry, University of Umea, Umea, Sweden

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**Abstract**

Polysomes from the frontal cortices of individuals who had histopathologically confirmed Alzheimer's disease were compared with polysomes from individuals who exhibited no neuropathological conditions. The cytosolic polysome yield from Alzheimer's disease frontal cortex was reduced 40% compared with that obtained from control frontal cortex. The translational activity per unit polysome of the Alzheimer's disease polysomes was only 50% of control in a reticulocyte lysate in vitro translation assay in which human polysomes do not undergo reinitiation. These differences exhibited brain region specificity in that polysomes isolated from Alzheimer's disease cerebellum were not different from control cerebellar polysomes. Thus, the disruptions are not due to a secondary and general response of the entire brain to the disease. These reductions were reflected by similar decreases in the translation of the mRNA for high molecular weight neurofilament polypeptide. Thus, the inhibition of polysomal mRNA translation is a mechanism by which gene expression is impaired in pathologically involved brain regions of individuals afflicted by Alzheimer's disease.

**Author Keywords:** Translational control; Alzheimer's disease; Polysome; Protein synthesis Corresponding author. *Correspondence:* W.C. Wallace, Department of Psychiatry, Mount Sinai School of Medicine, 1 Gustave Levy Pl., , New York, NY 10029, , U.S.A.